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Ventilator-associated events . . . perhaps not the answer

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In the most recent issue of *JICS*, Dr Thomas has rightly argued that ventilator-associated pneumonia (VAP) can be difficult to diagnose and that efforts to limit ventilator-associated morbidity must extend beyond VAP prevention.[1] We share his concerns but are unconvinced that adopting the US Centers for Disease Control and Prevention National Healthcare Safety Network's (CDC NHSN) ventilator-associated event (VAE) surveillance system[2] is currently the right approach for our population.[3]

In Wales, there has been a national surveillance programme to identify episodes of VAP since 2008, originally using the Hospitals in Europe Link for Infection Control through Surveillance definitions (HELICS, classified as PN 1 to 5, according to radiological, systemic, pulmonary and microbiological criteria). We recently investigated triggers for initiating antibiotic therapy for suspected ventilator-associated respiratory tract infection (VARTI) and the implications of variation in clinical practice for VAP surveillance.[4] Among 282 invasively ventilated patients admitted to intensive care unit (ICU) for 48 h or more, 32 developed VARTI – the main features relating to sputum, inflammatory markers and radiography. Strikingly in less than 50% cases was chest radiography performed at time of diagnosis, precluding diagnosis of VAP according to HELICS although such episodes were associated with significantly prolonged mechanical ventilation and ICU stay.

However, applying the new CDC NHSN definitions, we found no overlap between VAE and suspected VARTI. Other authors have also described potential limitations to VAE surveillance. A ventilator-associated complication (VAC) is identified when there is sustained increase in FiO_2 or PEEP after a stable baseline of 48 h or more.[2] However, many patients do not meet the definition of VAC because fluctuations in FiO_2 and PEEP prevent them achieving a stable baseline.[5] Preference for PEEP level or ventilator mode, such as APRV, may affect whether a patient qualifies for the diagnosis of VAC. Furthermore, the level of agreement between episodes of VAC and VAP has been questioned; patients developing a VAC have a range of pathology which might include VAP but alternatively may represent fluid overload or ARDS.[6] Interventions required to prevent and treat heterogeneous pathology are likely to differ and it may be difficult to understand the contribution of different processes to outcomes associated with VAC.

There are distinctions between the diagnostic needs of the clinician at the bedside and the surveillance administrator – and arguably there is risk of disengagement with a quality improvement programme that does not emphasise clinical discrimination. Furthermore, although VAE data are highly objective, in a paper-based clinical environment, the workload associated with screening should not be underestimated. Appreciating a wider burden of ICU-acquired respiratory infection, our approach has not been to dispense with HELICS definitions of VAP at this stage, but – acknowledging the variation in performance of chest X-ray and subjectivity in interpretation – to supplement with an additional category 'PN0' where there is pulmonary, systemic and microbiological evidence of VARTI but without radiographic evidence. We anticipate a potential value for the ICU clinician but also hope to standardise reporting across our region.

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